```
### Status: Path 1 of [Dialog Information Services via Modem]

### Status: Initializing TCP/IP using (UseTelnetProto 1 ServiceID pto-dialog)
Trying 31060000009999...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
 ******** HHHHHHHH SSSSSSSS?
### Status: Signing onto Dialog
 ********
ENTER PASSWORD:
 ******** HHHHHHHH SSSSSSSS? ********
Welcome to DIALOG
### Status: Connected


Dialog level 02.12.60D

Last logoff:  03mar03 12:57:11
Logon file001 03mar03 16:44:20
KWIC is set to 50.
HILIGHT set on as '*'
* *  New CURRENT Year ranges installed    **

File   1:ERIC  1966-2003/Feb 25
       (c) format only 2003 The Dialog Corporation

      Set  Items  Description
      ---  -----  -----------
Cost is in DialUnits
?b 155
       03mar03 16:44:23 User259876 Session D472.1
            $0.28    0.081 DialUnits File1
     $0.28  Estimated cost File1
     $0.28  Estimated cost this search
     $0.28  Estimated total session cost   0.081 DialUnits


File 155:MEDLINE(R)  1966-2003/Feb W4
       (c) format only 2003 The Dialog Corp.

      Set  Items  Description
      ---  -----  -----------
?s (neurogenesis) and (brain and (spinal (w) cord))
           2793  NEUROGENESIS
         544178  BRAIN
         168683  SPINAL
         120578  CORD
          90040  SPINAL(W)CORD
      S1    83  (NEUROGENESIS) AND (BRAIN AND (SPINAL (W) CORD))
?s s1 and (adenovirus or vector)
             83  S1
          18076  ADENOVIRUS
          46202  VECTOR
      S2     0  S1 AND (ADENOVIRUS OR VECTOR)
?s s1 and (therapy or treatment)
             83  S1
        1911215  THERAPY
        1405118  TREATMENT
      S3     3  S1 AND (THERAPY OR TREATMENT)
?t s3/3,k/all

 3/3,K/1
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.
```

11102683 21146241 PMID: 11252267

Reorganization of the human central nervous system.

Schalow G; Zach G A

General physiology and biophysics (Slovakia) Oct 2000, 19 Suppl 1 p11-240, ISSN 0231-5882 Journal Code: 8400604

Document type: Journal Article; Review; Review Literature

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

... included (1) our measurements of phase and frequency coordination between the firings of alpha- and gamma-motoneurons and secondary muscle spindle afferents in the human *spinal* *cord* , (2) knowledge on CNS reorganization derived upon the improvement of the functions of the lesioned CNS in our patients in the short-term memory and...

... our measurements of the natural firing patterns of sets of identified single neurons in the human spinal premotor network and re-learned coordinated movements following *spinal* *cord* and *brain* lesions. *Therapy* induced cell proliferation, and maybe, *neurogenesis* seem to contribute to the host of structural changes during the process of re-learning of the lesioned CNS. So far, coordinated functions like movements could substantially be improved in every of the more than 100 patients with a CNS lesion by applying coordination dynamic *therapy*. As suggested by the data of our patients on re-learning, the human CNS seems to have a second integrative strategy for learning, re-learning...

... has also been observed by us in single motor unit firing patterns measured electromyographically, it seems possible to follow up therapeutic intervention in patients with *spinal* *cord* and *brain* lesions not only based on the activity levels and phases of motor programs during locomotion but also based on the physiologic and pathophysiologic firing patterns...

... measured directly by rhythmicity upon the patient performing rhythmic movements coordinated up to milliseconds. Since rhythmic dynamic, coordinated, stereotyped movements are mainly located in the *spinal* *cord* and only little supraspinal drive is necessary to initiate, maintain, and terminate them, rhythmic, dynamic, coordinated movements were used in *therapy* to enforce reorganization of the lesioned CNS by improving the self-organization and relative coordination of spinal oscillators (and their interactions with occasionally firing motoneurons) which became pathologic in their firing following CNS lesion. Paraparetic, tetraparetic *spinal* *cord* and *brain* -lesioned patients re-learned running and other movements by an oscillator formation and coordination dynamic *therapy*. Our development in neurorehabilitation is in accordance with those of theoretical and computational neurosciences which deal with the self-organization of neuronal networks. In particular...

... the potential function of the integrated CNS activity, the change in self-organization becomes understandable. Movement patterns re-learned by oscillator formation and coordination dynamic *therapy* evolve from reorganization and regeneration of the lesioned CNS by cooperative and competitive interplay between intrinsic coordination dynamics, extrinsic *therapy* related inputs with physiologic re-afferent input, including intention, motivation, supervised learning, interpersonal coordination, and genetic constraints including *neurogenesis*. (ABSTRA

3/3,K/2

DIALOG(R)File 155:MEDLINE(R)

10862058 20415150 PMID: 10959248

Neuronal reorganization through oscillator formation training in patients with CNS lesions.

Schalow G; Zach G A
Department of Clinical Research, Swiss Paraplegic Centre Nottwil, Switzerland.
Journal of the peripheral nervous system : JPNS (UNITED STATES) 1998, 3 (3) p165-88, ISSN 1085-9489 Journal Code: 9704532
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... oscillatory firing has also been observed in electromyographic (EMG) single motor unit firing patterns, it seems possible to follow up therapeutic intervention in patients with *spinal* *cord* lesion not only based on the activity levels and phases of motor programs during locomotion but also based on the physiologic and pathophysiologic firing patterns and recruitment of spinal oscillators. Since rhythmic, dynamic, stereotyped, symmetric movements are mainly located in the *spinal* *cord* and only little supraspinal drive is necessary to initiate, maintain (especially), and terminate them, rhythm training methods were used to enforce reorganization of the CNS following *spinal* *cord* and CNS lesions to improve the self-organization and relative coordination of spinal oscillators which became pathologic in their firing following CNS lesion. Paraparetic, tetraparetic and *brain*-lesioned patients relearned running and other movements by an oscillator formation training. This development in neurorehabilitation is in accordance with those of theoretical and computational...

... and competitive interplay between intrinsic coordination dynamics, extrinsic training-related inputs with physiologic re-afferent input, including intention and supervised learning, and genetic constraints including *neurogenesis*.
Descriptors: Central Nervous System Diseases--physiopathology--PP; *Central Nervous System Diseases--*therapy*--TH; *Neurology--methods--MT; *Neuronal Plasticity

3/3,K/3
DIALOG(R)File 155:MEDLINE(R)

04938180 86011655 PMID: 4046073
Development of mouse *spinal* *cord* in tissue culture: IV. Effects of embryonic extracts on neuron formation and migration.
Houle J D; Fedoroff S
Journal of neuroscience research (UNITED STATES) 1985, 14 (2) p187-96, ISSN 0360-4012 Journal Code: 7600111
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Development of mouse *spinal* *cord* in tissue culture: IV. Effects of embryonic extracts on neuron formation and migration.
Possible influences upon patterns of *neurogenesis* expressed in vitro were examined quantitatively by the use of microfragment cultures of embryonic day 10 mouse neural tube. Crude extracts were prepared either from...

... outgrowth zones surrounding individual microfragments were reduced in area (indicating restricted neuronal migration) and in number of neurons present (indicating restricted production of neurons) following *treatment* with either of the extracts. The severity of reductions observed were related to the developmental age of embryonic tissue used for preparing the extract, as greatest reduction resulted from addition of embryonic day 18 *brain* extracts and to concentration employed, higher doses further restricting neuronal outgrowth. By increasing the concentrations of extract

. the proportional number of large-sized neurons forming...

... addition of embryonic mouse extracts to the medium. We propose that an endogenous negative feedback mechanism may be involved in the coordination of patterns of *neurogenesis*.
Descriptors: Neurons--physiology--PH; **Spinal* *Cord* --growth and development--GD; *Tissue Culture; *Tissue Extracts--pharmacology--PD; Astrocytes--physiology--PH; *Brain*; Cell Count; Embryo; Mice; Neurons --drug effects--DE; *Spinal* *Cord*--cytology--CY; Time Factors; Tissue Culture--methods--MT
?ds

```
Set     Items   Description
S1         83   (NEUROGENESIS) AND (BRAIN AND (SPINAL (W) CORD))
S2          0   S1 AND (ADENOVIRUS OR VECTOR)
S3          3   S1 AND (THERAPY OR TREATMENT)
?s (neurodegenerative (w) disease) and (gene (w) therapy)
            9694  NEURODEGENERATIVE
         1378763  DISEASE
            1262  NEURODEGENERATIVE(W)DISEASE
          605210  GENE
         1911215  THERAPY
           20157  GENE(W)THERAPY
      S4      27  (NEURODEGENERATIVE (W) DISEASE) AND (GENE (W) THERAPY)
?s s4 and (neurogenesis)
              27  S4
            2793  NEUROGENESIS
      S5       0  S4 AND (NEUROGENESIS)
?s s4 and (neurotrophic (w) factor?)
              27  S4
            9015  NEUROTROPHIC
         1897271  FACTOR?
            6746  NEUROTROPHIC(W)FACTOR?
      S6       7  S4 AND (NEUROTROPHIC (W) FACTOR?)
?t s6/3,k/all
```

6/3,K/1

DIALOG(R)File 155:MEDLINE(R)

14141665 22364836 PMID: 12477256

Neuroprotective *gene* *therapy* for Parkinson's disease.

Tenenbaum L; Chtarto A; Lehtonen E; Blum D; Baekelandt V; Velu T; Brotchi J; Levivier M; et al
Laboratory of Experimental Neurosurgery, Institut de Recherche Interdisciplinaire en Biologie Humaine et Moleculaire, Universite Libre de Bruxelles, Hjpital Erasme, 808, Route de Lennik, B-1070 Brussels, Belgium,. litenenb@ulb.ac.be

Current gene therapy (Netherlands) Dec 2002, 2 (4) p451-83, ISSN 1566-5232 Journal Code: 101125446
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: In Process

Neuroprotective *gene* *therapy* for Parkinson's disease.

Parkinson's disease (PD) is a *neurodegenerative* *disease* characterised by a progressive loss of the dopaminergic neurones in the substantia nigra pars compacta. Accumulating evidence indicates that apoptosis contributes to neuronal cell death...
... are poorly understood, neuroprotection can be achieved by interfering with neuronal cell death either directly or by preventing neuronal dysfunction. Potential agents for neuroprotection are *neurotrophic* *factors* , inhibitors of apoptosis or anti-oxidative agents. However, the existence of the blood-brain barrier precludes systemic delivery of these factors. In situ gene delivery...

... vectors as well as genetic elements with tightly controlled gene expression. Various relevant animal models for Parkinson's disease are available for the evaluation of *gene* *therapy* strategies. These include induction of cell death in specific neurone population through administration of toxins either directly in the brain or systemically, as well as...

6/3,K/2
DIALOG(R)File 155:MEDLINE(R)

12818842 21548870 PMID: 11690619
Sustained delivery of GDNF: towards a treatment for Parkinson's disease.
Zurn A D; Widmer H R; Aebischer P
Division of Surgical Research and Gene Therapy Center, Pavillon 4, CHUV, CH-1011, Lausanne, Switzerland. anne.zurn@chuv.hospvd.ch
Brain research. Brain research reviews (Netherlands) Oct 2001, 36 (2-3) p222-9, ISSN 0165-0173 Journal Code: 8908638
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Parkinson's disease (PD) is a *neurodegenerative* *disease* characterized by the progressive loss of nigral dopaminergic neurons. Although symptomatic therapies to substitute for the missing neurotransmitter dopamine are efficient at the early stages of the disease, the goal is to find alternative therapies which could protect dopaminergic neurons from the degenerative process. We have used two distinct *gene* *therapy* approaches to deliver the neuroprotective molecule glial cell line-derived *neurotrophic* *factor* (GDNF) in animal models of the disease: (i) an encapsulated genetically engineered cell line releasing GDNF (ex vivo *gene* *therapy*); and (ii) a lentiviral vector encoding the GDNF gene (in vivo *gene* *therapy*). Both approaches allowed protection of nigral dopaminergic neurons against lesion-induced cell death in rodent as well as monkey models of PD. Behavioral symptoms were...
Descriptors: Brain Tissue Transplantation--methods--MT; **Gene* *Therapy* --methods--MT; *Genetic Vectors--therapeutic use--TU; *Nerve Tissue Proteins--therapeutic use--TU; *Parkinsonian Disorders--genetics--GE; *Parkinsonian Disorders--therapy--TH; *Substantia Nigra--surgery--SU; Cells, Cultured; Diffusion Chambers, Culture--methods--MT; *Gene* *Therapy* --instrumentation--IS; Nerve Tissue Proteins--genetics--GE; Nerve Tissue Proteins--secretion--SE; Parkinsonian Disorders--physiopathology--PP; Substantia Nigra--pathology--PA; Substantia Nigra--physiopathology--PP
Chemical Name: Genetic Vectors; Nerve Tissue Proteins; glial cell-line derived *neurotrophic* *factor*

6/3,K/3
DIALOG(R)File 155:MEDLINE(R)

10876028 20435204 PMID: 10978846
Glial cell line-derived *neurotrophic* *factor* (GDNF) as a defensive molecule for *neurodegenerative* *disease* : a tribute to the studies of antonia vernadakis on neuronal-glial interactions.
Bohn M C; Kozlowski D A; Connor B
Children's Memorial Institute for Education and Research, Department of Pediatrics, Children's Memorial Hospital, Northwestern University Medical School, Chicago, IL 60613, USA. m-bohn@nwu.edu
International journal of developmental neuroscience : the official journal of the International Society for Developmental Neuroscience (ENGLAND) Nov 2000, 18 (7) p679-84, ISSN 0736-5748 Journal Code: 8401784

Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Glial cell line-derived *neurotrophic* *factor* (GDNF) as a defensive molecule for *neurodegenerative* *disease* : a tribute to the studies of antonia vernadakis on neuronal-glial interactions.

Research stemming from interests in neuronal-glial interactions has led to the identification of a number of novel trophic factors, such as the dopaminergic *neurotrophic* *factor* glial cell line-derived *neurotrophic* *factor* (GDNF). Delivery of the GDNF gene to rat models of Parkinson's disease suggests a potential clinical use of GDNF *gene* *therapy* for humans with this disease. This review article briefly summarizes the history of GDNF and the effects of GDNF gene delivery prior to or after...

Chemical Name: Nerve Growth Factors; Nerve Tissue Proteins; glial cell-line derived *neurotrophic* *factor*

6/3,K/4
DIALOG(R)File 155:MEDLINE(R)

10844203 20393182 PMID: 10933972

Parkinson's disease: a *neurodegenerative* *disease* particularly amenable to *gene* *therapy*.

Bohn M C

Children's Memorial Institute for Education and Research, Northwestern University Medical School, Chicago, Illinois 60614, USA. m-bohn@nwu.edu

Molecular therapy : the journal of the American Society of Gene Therapy (UNITED STATES) Jun 2000, 1 (6) p494-6, ISSN 1525-0016
Journal Code: 100890581
Contract/Grant No.: NS31957; NS; NINDS; NS39267; NS; NINDS
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Parkinson's disease: a *neurodegenerative* *disease* particularly amenable to *gene* *therapy*.

Descriptors: *Gene* *Therapy*; *Parkinson Disease--therapy--TH; Brain Stem--physiopathology--PP; Dopamine--genetics--GE; Dopamine--physiology --PH; *Gene* *Therapy*--methods--MT; Nerve Tissue Proteins--genetics--GE; Nerve Tissue Proteins--physiology--PH; Parkinson Disease--genetics--GE; Parkinson Disease--physiopathology--PP; Parkinsonian Disorders--therapy --TH; Prosencephalon...

Chemical Name: Nerve Tissue Proteins; glial cell-line derived *neurotrophic* *factor*; Dopamine

6/3,K/5
DIALOG(R)File 155:MEDLINE(R)

10259966 99233150 PMID: 10218782

Long-term actions of vector-derived nerve growth factor or brain-derived *neurotrophic* *factor* on choline acetyltransferase and Trk receptor levels in the adult rat basal forebrain.

Klein R L; Muir D; King M A; Peel A L; Zolotukhin S; Moller J C; Kruttgen A; Heymach J V; Muzyczka N; Meyer E M

Department of Pharmacology and Therapeutics, University of Florida, Gainesville 32610, USA.

Neuroscience (UNITED STATES) Mar 1999, 90 (3) p815-21, ISSN 0306-4522 Journal Code: 7605074
Contract/Grant No.: GM 35723; GM; NIGMS; HL 53665; HL; NHLBI; HL/DK 50257 ; HL; NHLBI; +

Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Long-term actions of vector-derived nerve growth factor or brain-derived *neurotrophic* *factor* on choline acetyltransferase and Trk receptor levels in the adult rat basal forebrain.

Trophic factor *gene* *therapy* may provide a rational treatment strategy for *neurodegenerative* *disease* . Recombinant adeno-associated virus vectors, incorporating a neuron-specific promoter driving bicistronic expression of green fluorescent protein and either nerve growth factor or brain-derived *neurotrophic* *factor*, transduced 10,000-15,000 neurons in the medial septum for periods of at least six months. Both cholinergic and non-cholinergic neurons expressed green fluorescent protein. Nerve growth factor and brain-derived *neurotrophic* *factor* vectors produced up to 50% increases in immunohistochemical detection of the acetylcholine-synthesizing enzyme in septal neurons ipsilateral to the injection. Increased levels of this enzyme, choline acetyltransferase, persisted for six months with the brain-derived *neurotrophic* *factor* vector. The nerve growth factor vector increased Trk receptor immunoreactivity in a volume of brain exceeding that of the transduced cells. Counterstaining for the neuronal...

Descriptors: Brain-Derived *Neurotrophic* *Factor*--pharmacology--PD; *Choline O-Acetyltransferase--metabolism--ME; *Nerve Growth Factors --pharmacology--PD; *Prosencephalon--metabolism--ME; *Receptor Protein-Tyrosine Kinases--metabolism--ME; *Receptors, Nerve Growth Factor...
; Brain-Derived *Neurotrophic* *Factor*--genetics--GE; Dependovirus --genetics--GE; Gene Expression--physiology--PH; Genetic Vectors; Luminescent Proteins--genetics--GE; Nerve Growth Factors--genetics--GE; Rats; Rats, Sprague-Dawley; Receptor, Ciliary *Neurotrophic* *Factor*; Recombination, Genetic; Time Factors; Transgenes--genetics--GE

Chemical Name: Brain-Derived *Neurotrophic* *Factor*; Genetic Vectors; Luminescent Proteins; Nerve Growth Factors; Receptor, Ciliary *Neurotrophic* *Factor*; Receptors, Nerve Growth Factor; green fluorescent protein; Choline O-Acetyltransferase; Receptor Protein-Tyrosine Kinases

6/3,K/6
DIALOG(R)File 155:MEDLINE(R)

09426120 97303966 PMID: 9160251

Prevention of motoneuron death by adenovirus-mediated *neurotrophic* *factors*.

Gimenez y Ribotta M; Revah F; Pradier L; Loquet I; Mallet J; Privat A
INSERM U. 336, DPVSN, University of Montpellier, France.

Journal of neuroscience research (UNITED STATES) May 1 1997, 48 (3) p281-5, ISSN 0360-4012 Journal Code: 7600111
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Prevention of motoneuron death by adenovirus-mediated *neurotrophic* *factors*.

Amyotrophic lateral sclerosis (ALS) is a fatal *neurodegenerative* *disease* characterized by progressive loss of motoneurons, and has no effective treatment. Experimental studies in rodents have shown that motoneurons respond to a variety of molecules including brain-derived *neurotrophic* *factor* (BDNF). and the glial-cell line-derived *neurotrophic* *factor* (GDNF). Here we investigated the neuroprotective effect of these growth factors, encoded by an adenovirus, on the death of axotomized facial motoneurons in newborn rats. We used a new *gene* *therapy* strategy that involves gene transfer to motoneurons by intramuscular injection of an adenoviral vector, which is retrogradely transported from injected target muscle (Finiels et al...

Descriptors: Adenoviridae--genetics--GE; *Brain-Derived *Neurotrophic* *Factor*--genetics--GE; *Genetic Vectors; *Motor Neurons--physiology--PH; *Nerve Tissue Proteins--genetics--GE; *Neuroprotective Agents; Animals, Newborn; Axons--physiology--PH; Brain-Derived *Neurotrophic* *Factor* --pharmacology--PD; Cell Death--drug effects--DE; Denervation; Facial Nerve --cytology--CY; Facial Nerve--enzymology--EN; Gene Transfer Techniques; Motor Neurons--drug effects--DE; Motor...
Chemical Name: Brain-Derived *Neurotrophic* *Factor*; Genetic Vectors; Nerve Tissue Proteins; Neuroprotective Agents; glial cell-line derived *neurotrophic* *factor*; beta-Galactosidase

6/3,K/7
DIALOG(R)File 155:MEDLINE(R)

07923540 94057844 PMID: 8239296
Cells engineered to produce acetylcholine: therapeutic potential for Alzheimer's disease.
Fisher L J; Raymon H K; Gage F H
Department of Neurosciences, University of California, San Diego, La Jolla 92093-0627.
Annals of the New York Academy of Sciences (UNITED STATES) Sep 24 1993, 695 p278-84, ISSN 0077-8923 Journal Code: 7506858
Contract/Grant No.: AG 10435-02; AG; NIA
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... the cognitive deficits that occur. However, current systemic strategies have met with limited success. An alternative strategy, that has been pursued in animal models of *neurodegenerative* *disease*, is to augment neurotransmitter function within the brain through tissue transplantation. Such implants have an advantage over conventional drug therapies in that the cells can...
... precisely placed within compromised areas of the brain. We have pursued a strategy of designing cells, through the use of molecular biology techniques, to produce *neurotrophic* *factors* and neurotransmitters. Recently, we developed a primary fibroblast cell line that was genetically modified to express choline acetyltransferase (ChAT). In vitro, these cells produced and...
Descriptors: Acetylcholine--biosynthesis--BI; *Alzheimer Disease--therapy --TH; *Choline O-Acetyltransferase--metabolism--ME; **Gene* *Therapy*
?ds

Set	Items	Description
S1	83	(NEUROGENESIS) AND (BRAIN AND (SPINAL (W) CORD))
S2	0	S1 AND (ADENOVIRUS OR VECTOR)
S3	3	S1 AND (THERAPY OR TREATMENT)
S4	27	(NEURODEGENERATIVE (W) DISEASE) AND (GENE (W) THERAPY)
S5	0	S4 AND (NEUROGENESIS)
S6	7	S4 AND (NEUROTROPHIC (W) FACTOR?)

?logoff
03mar03 16:51:51 User259876 Session D472.2
$4.88 1.525 DialUnits File155
$2.10 10 Type(s) in Format 3
$2.10 10 Types
$6.98 Estimated cost File155
$1.86 TELNET
$8.84 Estimated cost this search
$9.12 Estimated total session cost 1.606 DialUnits

Status: Signed Off. (8 minutes)